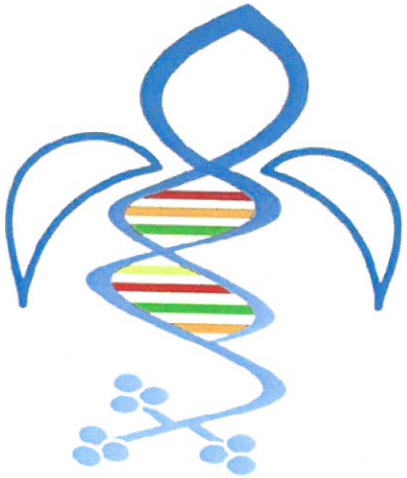
Arabidopsis thaliana PEN1 (AtSYP121) mediates triggering of innate immunity by bacterial lipo-oligosaccharides (LOS)

Erbs, Gitte; Dow, John Maxwell; Molinaro, Antonio; Newman, Mari-Anne

Publication date:
2009

Document version
Publisher's PDF, also known as Version of record



XIV
International Congress

On Molecular
Plant-Microbe Interactions

July 19-23, 2009, Quebec City, Canada

IS-MPMI 2009 XIV International Congress on Molecular Plant-Microbe Interactions Abstracts of Poster Presentations

DEPARTMENT OF PLANT BIOLOGY AND BIOTECHNOLOGY
FACULTY OF LIFE SCIENCES
UNIVERSITY OF COPENHAGEN

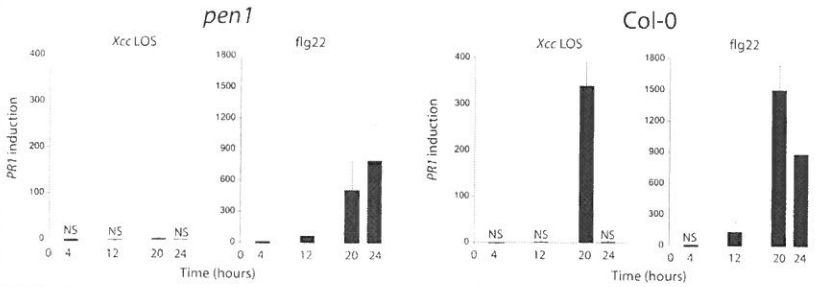
Arabidopsis thaliana PEN1 (AtSYP121) mediates triggering of innate immunity by bacterial lipo-oligosaccharides (LOS)

Gitte ERBS [1], John Maxwell DOW [2], Antonio MOLINARO [3] and Mari-Anne NEWMAN [1]

[1]Faculty of Life Sciences, Dept of Plant Biology & Biotechnology, University of Copenhagen, Denmark. [2]BIOMERIT Research Centre, Dept. of Microbiology, National University of Ireland, Cork, Ireland. [3]Dipartimento di Chimica Organica e Biochimica, Università di Napoli Federico II, Italy.

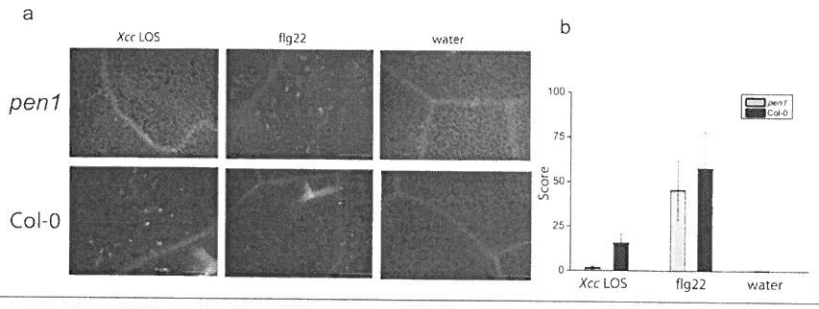
Abstract

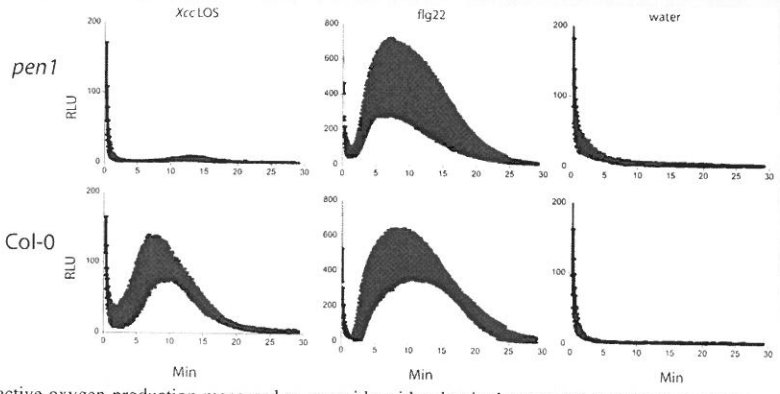
In eukaryotes, proteins of the soluble *N*-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) family are believed to be required for docking and fusion of intracellular transport vesicles with acceptor/target membranes. The *Arabidopsis* syntaxin PEN1 (*At*SYP121) is a SNARE protein that has been shown to play a role in pathogen resistance in *Arabidopsis* towards fungi (Kwon *et al.*, 2008). We present data to show that *Arabidopsis* PEN1 is also involved in signal transduction leading to the induction of the innate immune responses by particular microbe-associated molecular patterns (MAMPs) of bacterial origin. Specifically we show that PEN1 is required for induction of *PR1* gene induction, callose deposition and generation of reactive oxygen species (ROS) by LOS but not by flagellin.



Induction of defence responses in wild type and *pen1 Arabidopsis* by lipo-oligosaccharides (LOS) from *Xanthomonas campestris* pv. campestris (*Xcc*). Expression levels of *PR1* defence-related gene in response to either 50µg/ml LOS or 100nm flagellin (flg22) in *Arabidopsis pen1* mutant and *Arabidopsis* wild type leaves. Values are mean of three replicates ± SD of the fold up-regulation compared to water treated tissue, after normalization to 18SrRNA. P<0.001, NS: not significant. Three independent repetitions of each experiment were performed with similar results.

(**a**) Callose deposition in *Arabidopsis pen1* mutant and *Arabidopsis* wild type leaves 20 h after infiltration with LOS of *Xcc* or 100nM flg22; Light spots reveal callose fluorescence after leuco anililine blue staining. (**b**) Mean numbers ± SD of callose deposits per three fields of view in three replicate leaves from *Arabidopsis* plants. Scale bar: 200µM. Magnification X20. Water did not induce any of the tested defence responses.





Reactive oxygen production measured as peroxide with a luminol assay and expressed as relative light units (RLU) from *Arabidopsis pen1* mutant and *Arabidopsis* wild type leaves after exposure to 100µg/ml LOS from *Xcc* or 100nm flg22. Data are the means of at least three replicates ± SD.

Summary

Taken together our results suggest that PEN1 has a role in triggering immune responses in *Arabidopsis* in response to LOS but not in response to flg22. One explanation could be that PEN1 is required for the correct localisation at the plant cell plasma-membrane of the putative receptor(s) for LOS, but not for the flagellin receptor FLS2.

Alternatively, PEN1 may be required for endocytosis of an LOS complex, which may allow signalling to cytoplasmically located proteins to trigger defence responses.

Although we favour a model in which PEN1 is involved in exocytosis required for *Xcc* LOS triggered immunity in *Arabidopsis*, we cannot discount an alternate role in endocytosis. Importantly our findings indicate that PEN1 may have roles in plant disease resistance that have not been appreciated thus far.

References: Kwon, *et al.* (2008). Co-option of a default secretory pathway for plant immune responses. Nature 451, 835-840.